Convulsion of the lung: an historical analysis of the cause of Dr Johnson's fatal emphysema

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Cough: a convulsion of the lungs vellicated by some sharp serosity; pronounced coff[1].

Introduction

That Samuel Johnson, the Colossus of literature[2] died of emphysema complicated by cor pulmonale will not be doubted by anyone familiar with the observations at his necropsy. It has not, however, been sufficiently considered why the Great Lexicographer, who neither smoked nor engaged in an industrial occupation, developed this lethal affliction. In undertaking an analysis of the cause of Johnson's emphysema, it should be understood that diagnosis of pulmonary disease was primitive in the eighteenth century, and his necropsy, perfunctory by modern standards: one is therefore left with the compensatory expedient of reconstructing the nature and cause of his illness from a historical record that is both imperfect and incomplete, and in which the signification of terms differs from contemporary usage.

That this re-examination of the historical record is merited will be questioned. Votaries of Johnson will doubtless find its conclusions of interest, and among non-devotees, one hopes to stir gainful regard. Raleigh's assessment goes far in explaining this enduring reverence:

> This is the greatness of Johnson, that he is greater than his works. He thought of himself as a man, not as an author; and of literature as a means, not as an end in itself. Duties and friendships and charities were more to him than fame and honour . . . 'Books without the knowledge of life are useless; for what should books teach but the art of living'[3] (p 31), [and] He has almost become the tutelary genius of the English people. He embodies all that we most admire in ourselves. When we pretend to laugh at our national character, we call it John Bull; when we wish to glorify it, we call it Samuel Johnson[3] (p 32).

An indication of the remarkable length of duration and continuance of esteem in this extraordinary personage is the existence of a periodically updated bibliography[4] which cites 4282 critical studies in the 1970 edition, and acknowledges the incomplete state of this exhaustive compendium because of the absence of records of many eighteenth century publications.

Historical review

In 1849, Dr Squibb provided the first exclusively medical account of Dr Johnson's life in a paper read before the Harveian Society of London[5], in which he acknowledged the 'kind permission and liberality' of Dr James Arthur Wilson in permitting him to see the manuscript of the post-mortem examination that his father, James Wilson, had performed on Samuel Johnson in 1784[6]. Sir Humphrey Rolleston received this account from Lee Dickinson, the prosector's great-grandson by marriage in 1891, and quoted from it in the presidential address given before the Royal Medico-Chirurgical Society of Glasgow in 1924[7]:

> Wednesday, Dec. 15th, 1784, opened the body of Dr. Samuel Johnson for M. Cruikshank, in the presence of Dr. Heberden, Brocklesby, Butter . . . On opening into the cavity of the chest, the lungs did not collapse as they usually do when the air is admitted, but remained distended, as if they had lost the power of contraction; the air-cells on the surface of the lungs were also very much enlarged; the right lobe adhered very strongly to the diaphragm; the internal surface of the trachea was somewhat inflamed; no water was found in the cavity of the thorax. The heart was exceedingly large and strong . . .

Dr Wilson, 19 years of age at the time of the dissection, classified Johnson's disorder under the heading, 'asthma'. However, it has the pathological features of emphysema complicated by cor pulmonale; and it appears to provide the basis of the first pathological report of this disease[8], having preceeded Laennec's description[9] by 16 years. Baillie's account of this condition[8], given below, was based on the lung he obtained from the person who had requested the necropsy[7], Dr William Cruickshank, referred to above, one of four physicians attending Dr Johnson, without charge, during his last illness[2] (p 575).

> The lungs are sometimes, although I believe very rarely, formed into pretty large cells, so as to resemble somewhat the lungs of an amphibious animal. Of this I have now seen three instances. The enlargement of the cells cannot well be supposed to arise from any other cause, than the air not being allowed the common free egress from the lungs, and therefore accumulating in them. It is not improbable also that this accumulation may sometimes break down two or three contiguous cells into one and thereby form a cell of very large size.

Regrettably, dissection of the lung was not undertaken and the specimen no longer exists (E Allen, H Attwood, personal communications 1992). Moreover, Attwood[10] argues persuasively that the engraving in Baillie's *Morbid Anatomy*[9] (Fasc 2, Plate 6, Fig. 1), taken to represent Dr Johnson's lung, does not correspond to the description given above and, in fact, represents a lung from an individual who suffered from fibrosing alveolitis. One is therefore left with the sole recourse of attempting to reconstruct, principally

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from epistolary sources, the cause of Johnson's emphysema, which, as Larson points out, cannot be ascribed to tobacco smoke[11].

That Dr Johnson suffered from ill health since infancy is attested to in his autobiographical sketches and recounted in some detail by Rolleston[7], who indicates that he had unilateral blindness due to a corneal leukoma resulting from tuberculous keratitis. He was one of 200 individuals suffering from The King's Evil touched by Queen Anne at St James's Palace in 1712. Profound near-sightedness of the remaining functional eye is said to have accounted for his disinterest in the visual and performing arts[7]. He suffered from periods of melancholy so profound that he feared that madness would ensue[12]. In 1778, Johnson wrote his friend Dr Edmund Hector, 'My health has been, since my twentieth year, such as has seldom afforded me a single day of ease'. His respiratory health until the winter of 1755 appeared excellent as McHenry reports[13]

His ultimately fatal ailment, of which a minute account will be naturally desired, began shortly after the publication of his prodigious work, the three volume *Dictionary of the English Language.*

Meanwhile, during the lonely Christmas season he became ill with a severe case of bronchitis, possibly even pneumonia. Johnson described the illness to a friend, Robert Paul, on Dec. 29, 'I have never been out of doors since you saw me. On the day after I had been with you I was sore with a hoarseness which still continues; I had a cough so violent that I once fainted under its convulsions. I was afraid of my lungs. My physician bled me yesterday and the day before, first against his will, but the next day without any dissent. I had been bled once before, so that I have lost 54 ounces. I live on broaths and my cough, thank God, is much abated, so that I can sleep[12].

Succeeding correspondence frequently refers to episodes of severe and persistent winter bronchitis, remitting in the spring with the coming of warmer weather.

In 1762, Johnson, then 53, summered at the estate of his good friend Sir Joshua Reynolds, the same to whom Boswell dedicated his famous biography in 1791. Johnson's bronchitis had remitted and his respiratory status was excellent as the following anecdote, related by Miss Reynolds[13] (p 278) attests:

Before the house was a spacious lawn. When someone remarked that the lawn was ideal for racing, a young lady who was present boasted that she could outrun anyone there. At this, Johnson arose and said 'Madam, you cannot outrun me'.

The lady at first had the advantage; but Dr. Johnson, happening to have slippers on much too small for his feet, kicked them off up into the air, and ran . . . leaving the lady far behind him and . . . returned, leading her by the hand, with looks of high exultation and delight.

In the early 1760s Johnson wrote that he had been suffering from 'convulsions of the breast' since 1759[12]; 10 years later,

The last year has been wholly spent in a slow process of recovery. My days are easier, but the pertubation of my nights is very distressful . . . My lungs seem encumbered, and my breath fails me, if my strength is any unusual degree exerted, or my motion accelerated. I seem to bear exercise with more difficulty than in the last winter[14].

On the return from his annual trip to Lichfield in December 1773, he caught a cold . . .

he then developed a 'violent' fever which lasted six weeks. His cough too, grew more violent, harassing him almost incessantly day and night, finally becoming productive of sputum[12].

In February 1774 he wrote Boswell: 'I have been troubled for many weeks with a vexatious catarrh which is sometimes sufficently distressful'. In March of the same year: 'Of myself I can only add that having been afflicted many weeks with a troublesome cough, I am now recovered'. In July, to Langton[2] (p 232): 'I have never recovered from the last dreadful illness, but flatter myself that I grow gradually better: how much however, yet remains to mend'. In January 1776[12]: 'I have been afflicted through the whole Christmas with the general disorder of which the worst effect was a cough . . .'. McHenry observes

This bout of pulmonary disease lasted nearly four months. By March of 1776, with the help of warm weather, he was better. Johnson had had an attack of acute bronchitis probably complicated by pneumonia in view of high persistant fever. This illness was more severe than is at first apparent, for later he wrote that he had never cared for much walking since this illness in the winter of 1773-1774[12].

Three years later nocturnal dyspnoea and orthopnoea ensued, for which he received phlebotomies, physic, and opium. In 1780, he noted that

by spasms in the breast I was condemned to the torture of sleepyness without the power to sleep. These spasms after enduring more than twenty years I ease by three powerful remedies, abstinence, opium and mercury[15].

The same year he wrote Boswell,

I have this summer sometimes amended, and sometimes relapsed, but, upon the whole, have lost ground very much. My legs are extremely weak, and my breath is very short, and the water is now increasing upon me[15].

In 1783, 2 years before the publication of Withering's monograph on the foxglove, and 1 year before his death, he received squill for treatment of progressive dropsy: he passed 20 pints of urine, allowing him to leave his home after 4 months of confinement. After a brief respite, oedema reappeared, which, added to his dyspnoea, caused great suffering; sleep became possible only by the employment of opiates to relieve the latter. In December of 1784, during an exacerbation of his cough and dyspnoea, he asked Dr Brocklesby for a candid opinion of his case, and, being advised that he could not recover without a miracle, replied, 'Then . . . I will take no more physic, not even my opiates; for I have prayed that I may render up my soul to God unclouded'[2] (p 581). On 13 December 1784, at age 74, an effusion of blood from a self-inflicted scalpel wound in his calf, performed in a heroic but misguided effort to palliate his dyspnoea, precipitated his death from cardio-respiratory failure.

Discussion

Dr Johnson's death occurred nearly one-half century prior to Laënnec's recognition and description of bronchiectasis and introduction of diagnostic auscultation, some two centuries prior to the development

of imaging procedures capable of confirming his diagnosis during life, his post-mortem examination limited to superficies, and the specimen no longer existing, it remains to inquire what anamnestic criteria would prompt an experienced observer to a provisional diagnosis of bronchiectasis. A history of protracted and severe lower respiratory tract infection, followed by episodic recurrences of a similar nature, accompanied by expectoration of copious quantities of purulent sputum, terminating in respiratory failure and cor pulmonale, would suffice, since these were the characteristic features described by Laënnec in 1819[16] (pp 76-8):

Chapter VI. OF DILATATION OF THE BRONCHIA
The organic lesion which I am now to notice, seems to have been hitherto entirely overlooked, both by the anatomist and the practitioner. This oversight is easily accounted for by the circumstance that, as it generally occurs in a small portion of a bronchial tube, when observed, it has been mistaken for a larger branch. It can only be detected by tracing the individual bronchial tubes to their ultimate ramifications, - a thing which is rarely done in our examination of the lungs.

This species of dilatation is generally met with only in subjects that have died, after being affected by chronic catarrh. It is sometimes so considerable that the bronchial ramifications, which in their natural condition, would scarcely admit the point of a very fine probe, acquire the diameter of a goose-quill, or even of the finger. These dilated portions terminate in culs-de-sac capable of containing a hemp-seed, a cherry-stone, a filbert, or even an almond . . .

Case 18. A woman, 62 years of age, had been affected, ever since she was sixteen, with a disease of the chest, which exhibited most of the usual symptoms of consumption. The principal of these were - hemoptysis, very frequent, and renewed by the slightest causes; constant cough, with expectoration of opaque, yellow sputa, having sometimes the character of pus, sometimes of a puriform mucus; and respiration more or less impeded. These symptoms varied much; they had very marked remissions, but scarcely ever any positive intermission: they never prevented her from following her occupation of teaching the piano-forte. Her chest was well formed, and she had nothing of the consumptive configuration. Without any material aggravation of the pectoral affection, this woman became universally anasarcous, and died shortly after entering the hospital.

The history of Dr Johnson's ultimately fatal illness conforms closely to the clinical characteristics of this disease provided by Laënnec, with one omission: the character of the phlegm or catarrh is never described. Free of unusual respiratory problems until the age of 46, Dr Johnson developed a severe and prolonged pulmonary ailment in 1755, followed by episodes of protracted winter bronchitis. A decade later he developed persistent dyspnoea, which, after severe exacerbation in 1773-1774, became both more pronounced and persistent. Two years before his death in 1784, he developed profound peripheral oedema. Postmortem examination showed advanced emphysema, cor pulmonale, and a dense adhesion of one lung to the diaphragmatic pleural surface. The lungs were not dissected.

The heralding and constant feature of Johnson's illness was a violent, prolonged, and pernicious cough, exacerbating in winter, remitting in summer, sometimes accompanied by fever and phlegm. His choice of term, 'convulsion', in defining 'cough', considering his remarkable linguistic precision, is revealing; nor is it likely that its severity was exaggerated, for he regarded the display of personal accounts of suffering distasteful.

Dear Doctor (said he one day to an acquaintance, who lamented the tender state of his inside), do not be like the spider, man; and spin conversation thus incessantly out of thy own bowels[13] (p 276).

In pursuing the inquiry that Johnson's emphysema may have been the consequence of bronchiectasis, it is instructive to inquire into coeval accounts of these disorders, which, since they precede the era of cigarette smoking to which nearly all instances of emphysema are presently imputed, may permit a clearer understanding of the genesis of the subject's illness.

Wilson, as noted above, classified Johnson's lungs as asthmatic. However, Laënnec, who undertook to define emphysema as a clinical-pathological entity, also recognized its association with bronchitis[16] (pp 81-95):

Before terminating this chapter, it may be useful to make a few observations on one symptom which many nosologists have erected into a distinct disease - I mean Asthma. This word, which properly signifies difficulty of breathing, has been as much misused, and has been made the cognomen of as many different diseases as any work in medicine. It has been proved by Corvisart that a great part of the diseases so denominated, are, in fact, diseases of the heart and large vessels; and every person accustomed to morbid dissection is now aware that the cases denominated humid or humoral asthma are simply examples of chronic catarrh.

Laënnec's cases 19-23, which provide an account of emphysema, are instructive: three of the five patients had a history of a severe respiratory infection early in life, commonly in infancy or childhood; the same proportion had cor pulmonale. Two evidenced an underlying infectious, as opposed to inflammatory process: localized pneumonitis, pleural adhesions, and/or bronchial dilatation. Case 23 may have had cystic bronchiectasis. The average age at death of the five reported cases was 42[16] (pp 76-8). It should be well considered - since these clinical features are not those one expect to encounter in emphysema - that cases which, in the absence of prominent findings of bronchial dilatation, Laënnec denominated emphysema, we should currently style emphysema consequent to cylindrical bronchiectasis.

It is not commonly appreciated that emphysema can arise as a consequence of bronchiectasis. Ogilvie[17] examined pulmonary specimens of 31 individuals who underwent resection for this disorder and found that

emphysema was noted in all specimens and varied from small patchy areas to extensive areas, even in specimens in which collapse appeared complete on gross inspection. This last observation was interesting and rather unexpected, as little or no mention of emphysematous areas was made in the literature and from accounts of 'atalectasis' one gained the impression that collapse is complete.

In a more detailed analysis, Reid[18] concluded that obliterative airways changes, and not dilatation, were the structural abnormality of greatest significance in bronchiectasis and bronchiolectasis. McLean[19] observed that the earliest lesions in centrilobular emphysema were an obliterative bronchiolitis and alveolar ductitis and hypothesized that the distending forces distal to this obstruction resulted in alveolar wall disruption. In contrast, Leopold and Gough[20],

in their classic article on the pathology of emphysema, published the same year, observed patent communications between the bronchioles and the emphysematous spaces in all lesions of centrilobular emphysema, and suggested an inflammatory mechanism for the alveolar loss. The 'supplying' bronchioles invariably demonstrated inflammatory changes but none was obliterative; narrowing was common but severe in only 12% nor was it invariably present. Thurlbeck[21] (p 392), referring to bronchiectasis, echoes Reid's conclusions:

> It should be reiterated at this point that it is not the dilated airways which produce airway obstruction in these patients. The site of airway obstruction is in the obliterated distal bronchi and bronchioles, and this is compounded by mucus plugs. [He concludes] ... Bronchiectasis (or, more precisely, its associated feature of obliterative airway disease) is occasionally the major source of chronic airway obstruction, and these patients are often misdiagnosed today[21] (p 21).

In a physiological assessment of the same issue, Cherniack and Carton[22] concluded,

> The pattern of physiologic dysfunction observed in bronchiectasis is quite similar to that occurring in chronic bronchitis and emphysema.

Two observations at Johnson's necropsy which help to define the nature of the process underlying his emphysema are deserving of comment: pleural changes, and right heart failure. Focal pleural thickening is a frequent finding in bronchiectasis: Ogilvie[17] noted 'gross pleural thickening', in 25 of 35 excised lobes, and Goodman[23] stated that 'The lobes in all cases were markedly adherent to surrounding structures in the chest'. One may reasonably infer one or more past episodes of suppurative infections from this finding. Cor pulmonale, attributable to the pulmonary hypertension resulting from numerous bronchial artery-pulmonary artery shunts that evolve in this disorder, frequently attends bronchiectasis; it was noted to be the cause of death in from 0-47% in five series summarized by Konietzko[24].

That Johnson's emphysema was caused by chronic bronchitis merits consideration. The poet Robert Southey vividly characterized London's atmosphere in 1808 as '... a compound of fen-fog, chimney smoke, smuts and pulverised horse-dung!'[25]. Brimblecombe's evaluation of London air pollution[26] - based on a mathematical model employing geologic estimates of coal sulphur content, the records of London coal imports, and maps indicating the dimensions of the city - indicates that sulphur dioxide levels rose to a plateau between 1690 and 1880, reaching an annual average value of 150 $\mu g\, m^{-3}$, twice current United States National Ambient Air Quality Standard[27] of 80 $\mu g\, m^{-3}$. Eighteenth century London ambient H_2SO_4 levels are not readily derivable from this figure because the ratio of the two is highly variable, depending as it does on atmospheric conditions, height of discharge, and presence of neutralizing levels of NH_4. H_2SO_4 comes into existence when SO_2 undergoes sequential oxidation and hydration, and is 10-20 times more potent than SO_2 as a respiratory irritant[27]. Also unknown are levels of black smoke and suspended particulate matter, believed to act synergistically with H_2SO_4 as respiratory irritants, exacerbating asthma and chronic bronchitis[27]. In contrast to the acute adverse health effects of SO_2 and other pollutants derived from fossil fuel combustion, a causal role in chronic bronchitis has been difficult to establish[27]; epidemiologic studies of workers chronically exposed to high levels of SO_2 have shown no clearcut evidence for chronic effects[28]. Oswald and Medvei[29] evaluated symptoms of respiratory disease in London clerical workers by means of a questionnaire in the early post-war period. They observed a surprisingly high prevalence (9.4%) of bronchitis, defined as habitual productive cough associated with disability from exacerbations and/or breathlessness, among nonsmokers compared to 15.9% among smokers. The clinical import of these observations cannot be assessed as neither radiographic nor spirometric analysis was undertaken; nor was any attempt made to assess the role of London air-pollution in the genesis of the bronchitis, although this was suggested as a possible cause. In summary, there is no credible historical or epidemiologic evidence to indicate that Johnson's emphysema arose as a consequence of urban air-pollution. Furthermore, one might anticipate that, were this its cause, the condition he suffered from would be commonly seen and therefore easily recognized. Boswell however makes no mention of others among Johnson's circle of close friends who had this disorder, and Johnson in his own inquiries found no parallel example.

Summary and conclusions

Of Johnson's fatal emphysema, it appears probable, on available historical and anatomic evidence, that it resulted from bronchiectasis, a diagnosis favoured by the pattern of illness: a protracted and severe respiratory infection succeeded by annual episodes of severe winter bronchitis, remitting in summer, and culminating in respiratory insufficiency; and by the findings of pleural adhesion and cor pulmonale at necropsy. That it resulted from chronic bronchitis is a proposition both plausible and irrefutable without the specimen.

> To be ignorant is painful; but it is dangerous to quiet our uneasiness by the delusive opiate of hasty persuasion[30].

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Letters to the Editor

Down's syndrome

We read Ann Gath's paper on Down's syndrome with great interest (May 1994 *JRSM*, p 276). There are a couple of points we would like to make that may be of relevance.

First, during the years prior to his death Professor Lejeune (acknowledged only in the introduction) was investigating the potential therapeutic benefits of nutritional manipulation in the management of Down's syndrome. This promising work seems to be largely unrecognized in the UK[1].

Secondly, children with Down's syndrome can be incapacitated by the reactions of other individuals to their appearance. In these cases, release of epicanthic folds, reduction of protruding tongue and prominent ear correction can be offered by the plastic and reconstructive surgeon as a relatively simple option.

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Erosive pustular dermatosis of the scalp secondary to synthetic fibre implantation

We read with interest the report by Kelly *et al.* (May 1994 *JRSM*, pp 291-2) describing extensive erythema, crusting and erosions of the scalp following synthetic fibre implantation as a treatment for androgenic alopecia. We have also treated a patient with similar clinical features following synthetic fibre implantation. The clinical features of these cases are in keeping with a diagnosis of erosive pustular dermatosis of the scalp. First described in 1979[1], this unusual clinical entity is characterized by pustules, erosions and crusts affecting predominantly the scalp. Prolonged exposure to sunlight in subjects with long standing physiological alopecia, local trauma[2] and surgical procedures[3], have each been described as possible predisposing factors. All these factors could be implicated in cases which occur secondary to synthetic fibre implantation. Response to therapy can be variable, but a combination of potent topical steroids and antibiotics often proves the most effective treatment in cases precipitated by local trauma or sun exposure[1,2]. Our case, which followed synthetic fibre implants, improved following this treatment combined with extraction of the synthetic fibres.

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